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Review

Obstetric malpractice litigation and cerebral palsy in term infants

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ABSTRACT

Despite the recognition by many researchers that cerebral palsy (CP) is rarely related to obstetric malpractice, there are many instances where obstetricians face litigation when a child is diagnosed with cerebral palsy following a difficult delivery. The aim of this paper is to review relevant research papers to aid practitioners involved in obstetric malpractice litigation. It is also prudent to question the feasibility of costs for long-term care for children with disabilities being met through the legal process.

1. Background

1.1. Malpractice litigation

Most parents expect to give birth to a healthy, normal baby and are deeply disappointed when their expectation is not fulfilled. Encouraged by earlier research suggesting that EFM (electronic fetal monitoring) in labour could help to deliver a baby in good condition,[1] many also believe that good perinatal outcome is exclusively dependent on a high standard of antenatal and delivery care, which is both the norm and their right.

Advances in medical care in recent decades have led to increased life expectancy for persons with severe CP. The expenses associated with optimal long-term care of children with CP, and the requirement for medical indemnity in order to practice medicine, means that the legal path becomes an option for compensation and financial support that is increasingly being pursued.

In recent years, the increase in obstetric litigation has led many conscientious doctors to leave this subspecialty, or restrict their work exclusively to gynaecology to reduce the possibility of being sued.[2] Even if the court finds that the practitioner was not negligent, the process of being sued causes a degree of stress and emotional trauma, such that the doctor may find it difficult to return to regular practice.[2]

Medical indemnity costs for insurance have soared, particularly in obstetrics. In Australia, obstetricians pay some of the highest premiums for medical indemnity insurance since they are associated with about 18% of the cost of claims, despite forming only 2% of the physician group.[3] Some USA insurers declined to renew policies for doctors who had prior claims against them. The Obama administration is working on "health care reform" and proposals stress the importance of "reining in medical malpractice claims".

1.2. Tort law

In Australia in July 2002, a committee lead by the Honourable Justice Ipp, was convened to report on comprehensive reforms to the law of negligence designed to reduce the cost of injury claims. The Health Care Liability Act 2001 (NSW) was superseded by the Civil Liability ACT 2002 (NSW) and was aimed at limiting the amount of compensation that could be awarded. The goal was to ensure sustainability of the medical indemnity insurance industry.[4]

The Act provides minimal criteria to be met for a claim of negligence to succeed in civil proceedings. In the case of medical negligence, the plaintiff's legal team must show that the doctor who owes the patient a duty of care has breached that duty of care and that, as a result, the patient has suffered injury. This restricts the patient's ability to make an unreasonable and fictitious claim against the doctor, unless they can find expert witnesses who will claim that each criterion has been fulfilled.

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Tort law offers some comfort in that claims may not stand up in court. However, tort law may not be an effective system for compensating injured patients since the court's decision is often dependent on legal strategy, the ability to find suitable expert witnesses and the physician's performance as a witness in court, resulting in a poor correlation between litigation success and negligence.[5] Furthermore the high costs of court proceedings means that the majority of claims are settled out of court, inadvertently encouraging litigation as a means of financially supporting the disabled.

2. The diagnosis of obstetric injury causing CP

It is appropriate to consider the process by which obstetric injury is diagnosed as the cause of CP.

CP is an umbrella term for "permanent disorders of the development of movement and posture, causing activity limitation, that are attributed to non-progressive disturbances that occurred in the developing fetal or infant brain".[6,7] Despite being a permanent and non-progressive brain lesion, the clinical signs can change over time as the child develops. Reviewing the child's progress is necessary before making a definitive diagnosis of CP, to exclude transient conditions.[8]

The cause of the disturbance is not defined, many are possible and may consist of a series of factors known as a causal pathway. If CP in a child is the result of a pathway in which intrapartum damage is a factor, then neonatal encephalopathy (NE) is always present. NE is defined as abnormal neurological findings on examination within 12–24 h after birth with signs of cortical dysfunction, which includes lethargy, stupor and seizures in severe cases.

Like CP, NE is an umbrella term without standard definition, and careful reading of the definition used by any author is essential. Used in a broader sense, it can result from several causes of which intrapartum hypoxic injury is only one, so its presence alone does not prove the occurrence of intrapartum injury. Nor does NE necessarily lead to permanent neurologic impairment, but its presence is a prerequisite to making the association between intrapartum injury and CP.

Although Karin Nelson found that the strongest predictor for the development of CP in the term infant was the presence and severity of NE,[9] the same paper also reported that only 6% of all CP born at term had exhibited NE. Thus the great majority of term CP cannot be the result of intrapartum injury. Hankins and Speer discussed the essential criteria, based on the International Consensus, needed to infer that an intrapartum hypoxic insult has caused moderate to severe NE, which then results in CP.[10]

The term intrapartum asphyxia should be avoided.[11] Clinically, it refers to peri-partum signs compatible with, but not specific to, hypoxic exposure without necessarily implying permanent injury. Semantically, asphyxia means cessation of the pulse as a result of hypoxia – implying death, if not permanent injury. Thus medical record entries of "intrapartum asphyxia" may be inappropriately taken as evidence of intrapartum hypoxia having caused permanent injury. It is worth noting that some degree of fetal hypoxia accompanies vaginal delivery and contributes to the stimulus to commence air breathing.

3. Risk factors and causation

In total Western Australian population CP data, almost 80% of CP occurs in term or near term infants.[12] In birth years 95–99, 65% of CP was born at term and a further 14.2% was born at 32–36 weeks. The frequency of cerebral palsy in term births is 1.5–2 per 1000 live births.[13]

There are many causal pathways to CP in the term or near term infant[13] several of which include NE (see Table 1).

Table 1
Causal pathways to CP involving NE.

Brain anomalies	Malformations, neuronal migration disorders, infections, thrombosis
Risk factors that increase vulnerability	Multiple births, fetal growth restriction
Placental problems	
Catastrophic intrapartum events	Prolapsed cord, haemorrhage

The outcomes following these different pathways vary from death to CP to normal development and the outcome for a particular fetus cannot be predicted. The challenge is that it has not been possible to definitively diagnose permanent cerebral injury occurring intrapartum.

The use of magnetic resonance imaging (MRI) is becoming increasingly important in recognizing patterns of injury. Cowan et al showed that 80% of term infants with NE (defined as alterations in tone, feeding ability and consciousness plus intrapartum or neonatal signs of hypoxia and/or compromise, but excluding those with major birth defects or seizures in isolation) had MRI findings within 2 weeks of birth consistent with acute hypoxic brain lesions.[14] Thus 4 out of 5 of this subset of NE are most likely to be the result of hypoxic ischaemic encephalopathy (HIE) acquired brain damage at or shortly after birth, showing that the clinical inference of HIE is frequently but not invariably correct. The inference is less robust for those with neonatal seizures alone. One third of this sample went on to develop CP, but neither their contribution to the total CP population nor whether the intrapartum insults were likely to be obstetrically preventable was reported.

3.1. Antepartum factors

Multiple gestation is associated with increased risk of CP, particularly in the presence of the antenatal death of a co-fetus.[15]

Vascular occlusive disease is an important cause of morbidity. Thrombi were frequently noted pathologically in the placentas of children with CP that were reviewed for litigation.[16] It was postulated that placental thrombi may embolize and reach the fetal brain, but further study was recommended.

In a clinicopathologic analysis where the outcome of CP was the focus for litigation, the clinical and pathological data of 158 medico-legal cases were consecutively reviewed.[17] The results confirmed the role of sentinel events and emphasized the importance of placental lesions that may either directly cause or decrease the threshold for brain injury.

TORCH infections (Toxoplasmosis, rubella, cytomegalovirus and herpes virus) acquired by the mother during pregnancy are capable of injuring the fetal or neonatal brain.[7] Evidence of intrauterine infection such as maternal fever, abdominal pain or malodorous liquor during labour, is a frequent precursor to low Apgar scores and encephalopathy in the first week of life.[18,19] The possibility of intrauterine exposure to infection must be considered even when the infant does not appear to have sepsis.

3.2. Intrapartum factors

The fetus has an adaptive circulatory mechanism that allows preservation of neuronal integrity even when there is interruption of placental blood flow.[20] In a prospective study Perlman found that renal injury was related to poor long-term neurologic outcome.[21] He suggests that a single marker of in utero stress gives little prognostic information, but a constellation of markers is of greater value in identifying infants at greatest risk for brain injury.[20]

There have been few good population studies of NE. The Western Australian case-control study by Badawi et al compared 164 term infants exhibiting moderate to severe NE (broadly defined) with 400 randomly selected controls.[22] There was no evidence of intrapartum hypoxia in over 70% of NE cases. The causes of NE were heterogeneous and most commenced in the antenatal period.

In the United Kingdom, Evans et al studied the relationship between NE and CP in a cohort of 57,159 consecutive term births.[23] NE with seizures followed by 4-limb CP was strongly associated with severe acidosis and renal dysfunction, suggestive of an acute intrapartum problem, although only a minority resulted from an identifiable obstetric event. As suggested by other researchers, they postulated that genetic factors may influence how infants react to severe hypoxia and acidosis.[24,25]

4. Consensus statements

Consensus statements, based on the best evidence available at the time, were developed to assist clinicians and researchers in determining whether an intrapartum asphyxia event was likely to have caused brain damage.

The consensus statement of the Australian and New Zealand Perinatal Societies was first published in MJA 1995.[26] It states that there is no clear evidence that obstetric technologies to avoid or reduce fetal distress, in particular EFM and caesarean section, have reduced the prevalence of CP. The Western Australian population data on CP frequency shows no decrease, in spite of increased use of EFM during labour and increased caesarean section rate (from 10% to over 30% in Australia over 30 years).[27]

The international consensus statement was published in the BMJ in 1999.[28] The recommendations are similar to the MJA consensus. It was drawn up by the International Cerebral Palsy Task Force, which comprised a wide range of clinicians and allied health practitioners. They note that it is difficult to retrospectively identify the antenatal causes of CP. The clinician might suspect hypoxic cerebral damage from a myriad of signs, but none are specific. The guidelines give 3 essential criteria, all of which are necessary before an acute intrapartum event sufficient to cause neurological impairment long term can be considered possible. 5 non-essential criteria increase the likelihood of this possibility. These criteria enable experts to retrospectively determine the likelihood of an intrapartum cause for CP. The consensus statement also lists factors, such as major congenital anomalies, antenatal infections and growth restriction, that suggest a cause for CP, other than acute intrapartum hypoxia.[28]

The type of CP helps when exploring aetiology. Researchers have shown that spastic quadriplegia and dyskinetic CP are the subtypes associated with acute intrapartum hypoxia.[29] However, neither is specific to intrapartum hypoxia.

The task force concluded that there is a body of research to indicate that in the majority of cases, CP is due to multi-factorial causes. More recent research suggests that MRI performed early in infants with NE might help to predict both cognitive and motor outcome.

5. Compensation and long term care

Learning that one's child has CP is distressing for parents, particularly when they realize that the condition is long-term and will significantly affect the development of their child, in all but very mild cases. The ongoing financial cost brings further stress. The Australian Medicare system provides for basic medical and allied health care, but the cost of regular therapy and education beyond the Medicare allowance is prohibitive for many families. The option of pursuing a legal path for financial compensation may be tempting, but is associated with yet more stress and significant delays to resolution.

The departure of practicing obstetricians following large payouts for medical malpractice in the past and budget blowouts for medical defense organizations, has resulted in great community disadvantage, particularly in rural areas, because cumulative years of obstetric experience and training have been lost[2] Furthermore, a significant proportion of the payouts cover the legal and court related expenses, leaving less available for patient care and services and the majority of disabled children are left without compensation.

A better system of compensation is required, if we believe that the disabled have a place in our society, whereby payments are made to all families with disabled children (regardless of questions of fault or negligence) throughout the child's life, with adjustments over time according to the child's changing needs. Rather than one large payout that could inadvertently be used in ways that may be of little benefit the child, payments over several years to assist with care, therapy and medical costs might allow targeted financial support for the child's needs. The ongoing payments could be tied in to regular review of the child's function and disability. Parents/caregivers would thereby be freed of the responsibility for managing large sums and the fear that mismanagement or long duration of life result in the person with CP outliving the funds required to maintain them. Funding for such a system will need careful thought and require contributions from Government, medical defense and insurance agencies.

This approach does not mean that negligent doctors are "let off the hook". Professional bodies or tribunals do play a part where discipline and/or education of the doctor is needed, allowing all to learn from any mistakes that have been made. The current Civil Procedure Rules requires that experts from both legal parties respond to a case conference directive from the court. The experts are required to discuss their opinions, to reach an agreement based on the best available evidence and provide a written report to the Court. This attempts to overcome expensive proceedings involving many disparate experts and reduces the amount of time spent in court. These systems should be used in preference to litigation, which is inequitable (only a small proportion of children benefit), inefficient and expensive as a means of funding disability. The process can be traumatic for litigants and defendants alike and discourages the culture of openness necessary to enable people to learn from mistakes. There may still be the need for civil court proceedings in some cases, but these would be driven by the desire to curtail repeated or willful negligence rather than to recoup medical expenses.

6. Conclusion

Due to the extensive work done by the International Task Force we now have criteria in the international consensus statement that can be applied by experts who provide opinion concerning the association between CP and intrapartum events.

There is still work to be done in relation to compensation for the long-term care of CP patients and others with severe/profound disabilities. This challenge, like that of the Task Force, requires consultation with various agencies, including government and insurance groups. It is encouraging to learn that the Australian Government is conducting a feasibility study into a National Disability Insurance Scheme, which will cover persons with disabilities irrespective of cause. Studdert et al discussed the options for malpractice reform and challenge us to consider the issues that need to be addressed.[30]

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